Treatment of Toxicodendron Dermatitis (Poison Ivy And Poison Oak)

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ABSTRACT

Toxicodendron dermatitis results from a reaction to an oil soluble oleoresin that is present in many parts of the poison ivy and poison oak plants. Prophylactic measures include avoidance, protective clothing, barrier creams and hyposensitization. Treatments include washing the area immediately with a solvent suitable for lipids and the use of antiinflammatory agents, especially corticosteroids.

Key Words: poison ivy, poison oak, Toxicodendron dermatitis

Poison-ivy dermatitis, the prototype allergic contact dermatitis, is so common in North America that physicians at all levels of training usually recognize it. Approximately 61% of persons in the United States are patch-test positive, and another 31% seem to become sensitive after they are exposed. The acute, often vesicular eczema typically appears in hand print and finger-mark patterns because it is often transferred from the hands to areas of skin that absorb more efficiently. This results in the formation of “streaks” of vesicles and sometimes bullae that are recognized by physicians as “poison ivy” (dermatitis). The allergy is to an oil soluble oleoresin present in tiny “resin” canals in many parts of the plant. Chemically the antigenic material comprises alk(en)yl catechols with predominantly pentadec(en)yl catechols in poison ivy and heptadec(en)yl catechols in both eastern and western poison oak. The mechanism is a classic delayed hypersensitivity-type allergic contact dermatitis mediated predominantly through the endogenous pathway by CD8+ T-cells. The basic mechanism is presented in two recent reviews.

Prophylactic measures for Toxicodendron dermatitis include avoidance, protective clothing, barrier creams, hyposensitization, and cleansing the affected area right away with soap and water or a solvent suitable for lipids. Anti-inflammatory agents, especially corticosteroids are used for treatment.

Prophylaxis

The most effective way to prevent contact dermatitis to poison ivy is avoidance of the plant, and this requires plant recognition in all four seasons. The method used for identification of poison ivy, poison oak and poison sumac is given in several published reviews. Several suggestions are illustrated in Figure 1. When one is in doubt as to whether or not a plant is a Toxicodendron, the black-spot test can be done by crushing sap from leaves onto a sheet of white paper, observing precautions. The resulting stain should darken on exposure to the air if it came from a Toxicodendron. However, this test employs only one quality of these plants, and should not be a substitute for other proved means of identification.

For persons at risk, protective clothing is recommended. Vinyl (PVC) gloves are especially useful, but rubber gloves allow penetration of the antigen. Contaminated fomites e.g., animal fur, garden tools and even clothing, can also transfer the antigen. If garments are only mildly to moderately soiled, they may be amenable to washing or better dry cleaning.

A number of barrier substances may reduce the severity of reactions from a standardized exposure. Examples include Ivy Block, quaternium-18 bentonite cream, or Stokogard cream. The last product contains multiple amino groups that are intended to prevent absorption by binding the antigen on the skin surface. It is applied...
prophylactically and washed off within a few hours. Ivy Block is an OTC preparation that has been approved by the U.S. Food and Drug Administration. It reduces experimental patch test reactions. A number of creams have some efficacy, but some products marketed for this purpose, while possessing some benefit, are somewhat less potent.

**Hyposensitization**

Oral and injectable urushiol has been administered to achieve a form of hyposensitization. Earlier investigators such as Shelmire were able to produce reduced reactivity with relatively crude preparations. This usually involved administration of progressively increasing doses of the causative antigen by mouth or by injection, over a period of many months. However, sometimes complications were severe. The degree of hyposensitization in modern times has usually been less dramatic, but still useful. The earlier reports of greater success probably were achieved at somewhat greater risk of complication, and today the antigens available are, for the most part, too weak to be effective at the dosages recommended. Interestingly, people who are exposed daily in their workplace tend to develop a form of individual tolerance called “hardening.” A classic example occurs in workers applying Japanese lacquer. It is also seen in patients treated for alopecia areata and other conditions where contact dermatitis is repeatedly produced therapeutically. The kits that were once used for oral hyposensitization are no longer available. The reason for such oral hyposensitization seems to be induced by anti-idiotypic antibodies.

**Figure 1:** Reprinted from Clinics in Dermatology, Volume 4(2), Guin JD, Beaman JH, Toxicodendrons of the United States, pp 137–48, 1986, with permission from Elsevier Science.

The urushiol from the plant is readily absorbed through the skin, so people who are highly allergic should try to remove the antigen within a few minutes of exposure. Soap and water are helpful, but any solvent suitable for lipids would likely be satisfactory, provided it is used immediately. Moderately sensitive people probably have a bit longer, about 30 minutes, to try to remove most of the antigen. When none of these is available, water alone can be beneficial.

Bathing with a contaminated hand can be a problem because of possible spread of the antigen, so some measure of common sense must be employed.

**Corticosteroid Treatment of Toxicodendron Dermatitis**
Oral therapy is usually effective if the patient is given an adequate dosage for a sufficient period of time. In an adult, one usually starts with 30-60mg of prednisone per day over 14–21 days.  

Treatment is normally continued for at least fourteen days after the initial exposure. Injectable (TM) corticosteroids are also effective, but they seem to have little advantage, and the repository forms do not allow day-to-day control. In some cases where systemic therapy is contraindicated or where distribution is limited, moderately potent topical corticosteroids can be used with 24-hour occlusion. This method uses category II to V topical corticosteroids under occlusion for at least two 24-hour applications, usually with a day of rest between. Absorption is more efficient using a 1–3 day application than it is for periods of less than 24 hours. Use of the less complex formulations such as a corticosteroid in petrolatum (only) may help reduce the increase in new allergies caused by the broadening of the allergic base (extended allergen syndrome). In very small areas intradermal triamcinolone acetonide or diacetate 2 mg/ml can be quite effective. Higher concentrations are usually unnecessary and in some areas may induce atrophy.  

Conclusion  

Poison Ivy dermatitis is better prevented than treated, and recognition of the plant is the best method of prevention. Barrier creams, protective clothing and especially disposable vinyl gloves are useful in reducing exposure. Treatment is with systemic corticosteroid therapy unless there is a contraindication. In the latter case, topical corticosteroids with 24-hour occlusion are beneficial.  

References  


